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To Estimate Level of Aspartate Aminotransferase Concentration of Saliva in Chronic Gingivitis and Chronic Periodontitis Patients Prior To and Following Non-Surgical Periodontal Therapy: A Clinico- Biochemical Study

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Abstract
Background: Saliva can be used as a diagnostic fluid in dentistry. Various enzymes have been proposed as markers
for periodontal destruction. One of them is aspartate aminotransferase, for which salivary analysis can offer a cost effective approach for monitoring the disease. Changes in enzymatic activity reflect metabolic changes in the gingiva and periodontium in inflammation. Aims: The purpose of this study was to assess the aspartate aminotransferase levels in saliva prior to and following scaling and root planning (SRP) at 1 month and 3 month interval and correlating it with the clinical parameters in generalized chronic gingivitis and chronic periodontitis patients. Materials and Methods: Thirty patients with generalized chronic gingivitis and 30 with generalized chronic periodontitis were selected. The activity of aspartate aminotransferase levels in saliva were assessed biochemically before and after SRP at 1 month and 3 months. The aspartate aminotransferase levels were correlated with clinical parameters (gingival index and probing depth). Statistical Analysis Used: A paired t test was done. Results: A decrease in gingival index, probing depth, and aspartate aminotransferase levels were seen in both the groups at 1 and 3 months which was found to be statistically highly significant (P value 0.00). Aspartate aminotransferase levels were statistically significantly correlated with the clinical parameters at baseline ($P < 0.05$) but at 3 months, a positive correlation was seen in both the groups which was statistically insignificant ($P > 0.05$). Conclusions: Elevated salivary aspartate aminotransferase levels were seen in generalized chronic gingivitis and chronic periodontitis patients, with higher values recorded in generalized chronic periodontitis correlating to the tissue destruction taking place in these conditions.

Key words: *Aspartate aminotransferase, diagnosis, periodontal destruction, saliva*

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I. Introduction

Gingivitis and periodontitis are inflammatory conditions of infectious nature, caused by a complex of anaerobic, Gram-negative bacteria. Periodontitis is a multifactorial disease which is affected by both genetic and environmental factors.[1] periodontitis is characterized by loss of connective tissue attachment and bone around the teeth in conjunction with the formation of periodontal pockets due to apical migration of the junctional epithelium· Bacterial virulence factors degradation of host tissues or cause the release of biologic mediators from host tissue cells that lead to host tissue destruction. Mediators produced as a part of host response that contribute to tissue destruction include proteinases, cytokines and prostaglandins. Also, a variety of enzymes produced by periodontal microorganism cause tissue destruction.[2]

Traditional diagnostic procedures used clinically include probing pocket depth and radiographic assessment of alveolar bone loss. Probing depth and attachment level measurements or evaluations from radiographs can only describe the past history of periodontal disease and do not reveal current or future disease

activity. Advances in oral and periodontal diagnostic research are moving toward methods by which periodontal disease can be identified and quantified by objective measures such as biomarkers.[3] diagnostic methods used for periodontal disease are clinical, biochemical, microbiological, immunological, and genetic.[4] The markers receiving the most attention in active periodontal lesions are aspartate aminotransferase (AST), collagenase, prostaglandin E2, Beta-glucoronidase, lactate dehydrogenase, arylsulfatase, and elastase. These enzymes are released from dead/dying cells, neutrophils, epithelial cells, and connective tissue at the affected sites[5]. AST, previously termed serum glutamic oxaloacetate transferase (SGOT), is one such marker of tissue destruction, suggested by its successful use as a diagnostic adjunct in human cardiac and hepatic tissue necrosis.[6]

Whole saliva is a mixture of the secretions of the major and minor salivary glands, mucosal transudations, gingival crevicular fluid, serum and blood derivatives from oral wounds, desquamated epithelial cells, expectorated bronchial and nasal secretions, bacteria and bacterial products, viruses and fungi, other cellular components, and food debris. It is a complex fluid containing an entire library of hormones, proteins, enzymes, antibodies, antimicrobial constituents, and cytokines.[7]

Nonsurgical periodontal therapy (NSPT) is the cornerstone of periodontal therapy and the first recommended approach to the control of periodontal infections. It is also known as "Cause related therapy, "Phase I therapy or Etiotrophic phase," and "Initial therapy." It is defined as "plaque removal, plaque control, supragingival and subgingival scaling root planing.[8] It has also been shown that SRP alone is effective in the complete elimination of pathogenic microbes in case of suprabony pocket and there is not effective alone in deep periodontal pockets that required addition adjuncts to antibiotic and periodontal surgical therapy.[9]

AST is a cytoplasmic enzyme, salivary AST levels in periodontitis are also related to the type of tissue affected by necrosis. AST is an enzyme normally confined to the cell, which is released to the gingival crevicular fluid and saliva upon cell death in the active phase of periodontal disease. Chambers et al., Imrey et al. and Yucetal-Tuncer et al. showed that increased levels of AST in crevicular fluid reflect active soft-tissue destruction in the periodontium in ligature-induced periodontitis.[10] Fibroblasts from the periodontal ligament produce significantly lower levels of AST than gingival epithelial cells. The increase in AST activity in periodontal disease is probably caused by cytolysis of periodontium cells and/or by gingival bleeding. Salivary AST could be used, among other salivary biochemical parameters, as a useful marker for monitoring periodontal disease.[11]

II. Material And Methods

Study design: randomized controlled clinical trial.

Subject selection criteria: A total number of 60 patients aged 20-65 years of both gender visiting the outpatient department of Government Dental College, Ahmedabad. An Informed consent was obtained from the patients and the ethical committee of the institute approved thise clinical trial. These patients were scrutinized for the inclusion and exclusion criteria. 30 patients in chronic generalized gingivitis and 30 patients chronic generalized periodontitis.

Study Location: study was done in Department of periodontology of government dental college and hospital with combined B. J. Medical College, civil hospital campus, asarwa, Ahmedabad, Gujarat.

Study Duration: April 2019 to April 2020

Sample size: 60 patients.

Group divisions

The selected patients were grouped as:

- Group I: 30 patients with generalized chronic gingivitis.
- Group II: 30 patients with generalized chronic periodontitis.

Criteria for group division

• Group I:- Plaque-associated gingivitis or marginal gingiva was erythematous and edematous with bleeding on probing. Sulcus depth was ≤3 mm. There was no clinical loss of attachment and gingival index score (Loe and Silness index, 1963) was ≥2.0.

• Group II: - Sites from patients diagnosed with chronic periodontitis with a probing depth of >5 mm.

Each group was assessed for the following clinical parameters at baseline, 1 month, and 3 months: Gingival Index (using Loe and Silness index 1963) and Pocket depth (using UNC-15 probe).

Inclusion criteria

1) Aged > 20 to 65 years.
2) Good general health.
3) A minimum of 20 teeth, and
4) Not undergone any dental treatment for the past 1 year.

5) No use of medications such as antibiotics, anticoagulants, steroids, hormonal therapy in the last 6 months.

Exclusion criteria

1) History of tobacco and alcohol use.
2) Infection disease.
3) Inflammation bowel disease.
4) Rheumatoid arthritis.
5) Hypertension.
6) Diabetes.
7) Organ transplantation, and
8) Cancer therapy.

Procedure methodology

Subjects were seated comfortably in a semireclined position on the dental chair and instructed to rinse mouth thoroughly with 15 ml of water. Subjects were then instructed to be seated for 5 minute and directed not to speak, eat/rinse and minimize Orofacial movements during the 5 min period. Subjects were instructed to spit the pooled unstimulated saliva (3ml) from the floor of the mouth into sterile plastic vials (Spitting method). The plastic vials were then closed and sent to the biochemistry lab for assaying the AST levels.

Scaling and Root Planing

After this, the subjects were subjected to through scaling and root planning and saliva sampling was done at 1 month and 3 months follow-up.

Biomarker analyses

Step 1:- Each saliva sample (3 ml) was pipetted into a clean microcap tube and clarified by centrifugation at 3000 rpm for 15 minutes.

Step 2:- The supernatant was transferred to clean microcap tubes and used immediately for a Semiautomatic Biochemistry Analyzer (TULIP Evolution 3000). Concentration of aspartate aminotransferase enzyme was determined using an SGOT Kit, according to the manufactures instruction. The results of aspartate aminotransferase assay were expressed as units/ litter for concentrations.

Biochemical estimation of AST levels

Biochemical assay for salivary AST levels was done using semi automatic analyzer (Protonic Proto 99). This analyzer works on optimal UV test as per IFCC (International federation of clinical chemistry and laboratory medicine) recommendations. AST formerly called glutamate oxaloacetate catalyzes the reversible transfer of an amino group from aspartate to α-ketoglutarate forming glutamate and oxaloacetate. The oxaloacetate produced is reduced to malate by malate dehydrogenase (MDH) and NADH. The oxidation of NADH is measured at 340 nm. The rate of decrease in the concentration of NADH measured photometrically is proportional to the catalytic concentration of AST present in saliva sample.

IFCC methodology

L- Aspartate + α ketoglutarate AST glutamate + oxaloacetate

Oxaloacetate + NADH + H^+ MDH malate + NAD^+

AST: Aspartate aminotransferase,
LDH: Lactate dehydrogenase
MDH: Malate dehydrogenase

Fig. 1: Gingival index scoring at baseline for group I

Fig. 2: probing pocket depth at baseline for group I

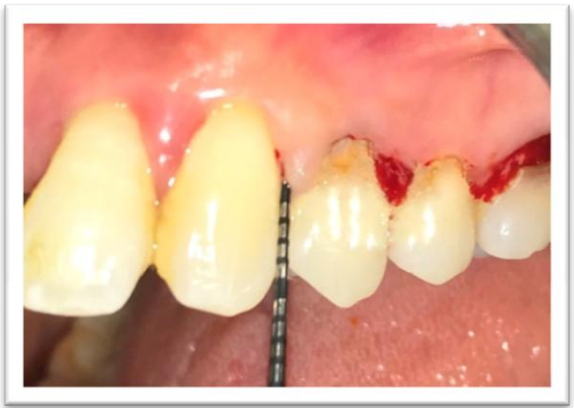


Fig.3: Gingival index scoring at baseline for group II

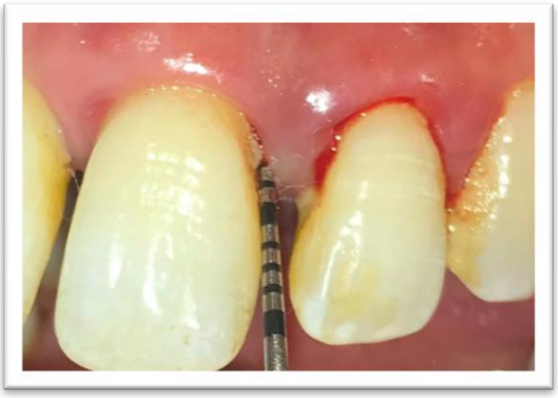


Fig.4: probing pocket depth at baseline for group II

III. Statistical analysis

The statistical significant of the difference in salivary levels of aspartate aminotransferase and all the clinical parameters in group I and group II were analysed using the paired t test. P value < 0.05 is statistically significant. Comparison between group I and group II for all the measures of periodontal parameters and AST levels were analyzed by applying Student paired "*t*" test. Karl Pearson's correlation coefficient ("*r*"). The magnitude or degree of relationship between two variables is called correlation coefficient and denoted by r.

1. r = 0 when there is no correlation between the two variables.
2. When there is complete relationship, the correlation coefficient is +1 or – 1.
3. If 0 < r < +1 then there is positive correlation and if – 1 < r < 0, then there is negative correlation.

Table No. 1. Demographic of study population.

Variables	Group A (n=30)	Group B (n=30)	p-value
Age (yrs)	37.7 ± 13.6	50.4 ± 9.10	< 0.0001*
Male	12 (40%)	13 (43.3%)	0.793 (NS)
Females	18 (60%)	17 (56.6%)	

One-way ANOVA test, statistically significant, NS - Non significant

Table No.2: Test of significance (paired't' test) among various parameters in Group I

Parameters	Mean±SD			P value
	Baseline	1 month	3 months	
Probing pocket depth (mm)	1.64±0.42	1.51±0.41	1.38±0.38	0.049
Gingival index scores	1.76±0.22	0.67±0.14	0.44±0.08	0.024
AST (unit/liter)	59.9 ± 8.10	46.8 ± 7.38	24.8 ± 6.73	0.084

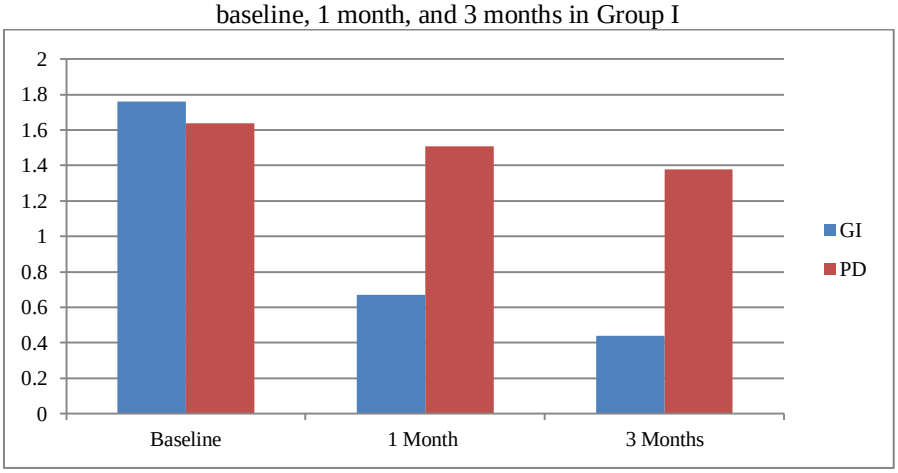
SD – Standard deviation; AST – Aspartate aminotransferase

Table No. 3: Test of significance (paired't' test) among various parameters in Group II

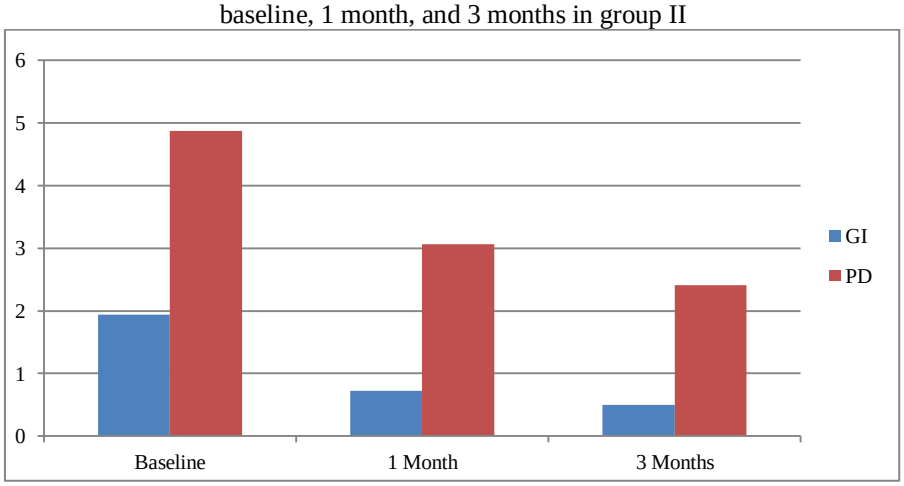
Parameters	Mean±SD			P value
	Baseline	1 month	3 months	
Probing pocket depth (mm)	4.87±0.33	3.06±0.54	2.41±0.37	0.029
Gingival index scores	1.94±0.40	0.72±0.16	0.50±0.12	0.021
AST (unit/liter)	81.0 ± 8.66	54.4 ± 7.37	29.7 ± 6.61	0.087

SD – Standard deviation; AST – Aspartate aminotransferase

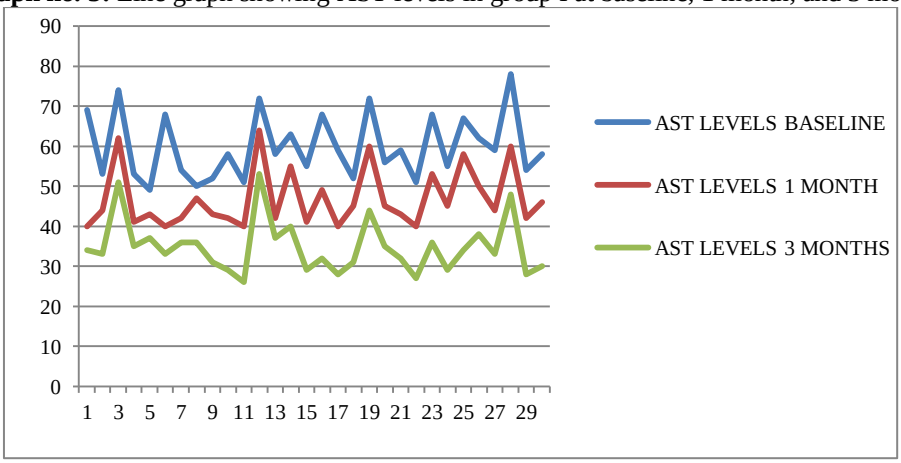
Graph no. 1: Comparison of clinical parameter (Gingival index and Probing depth) values of Mean with SD of baseline, 1 month, and 3 months in Group I



Graph no. 2: Comparison of clinical parameter (Gingival index and Probing depth) values of Mean with SD of baseline, 1 month, and 3 months in group II



Graph no. 3: Line graph showing AST levels in group I at baseline, 1 month, and 3 month



Graph no. 4: Line graph showing AST levels in group II at baseline, 1 month, and 3 month

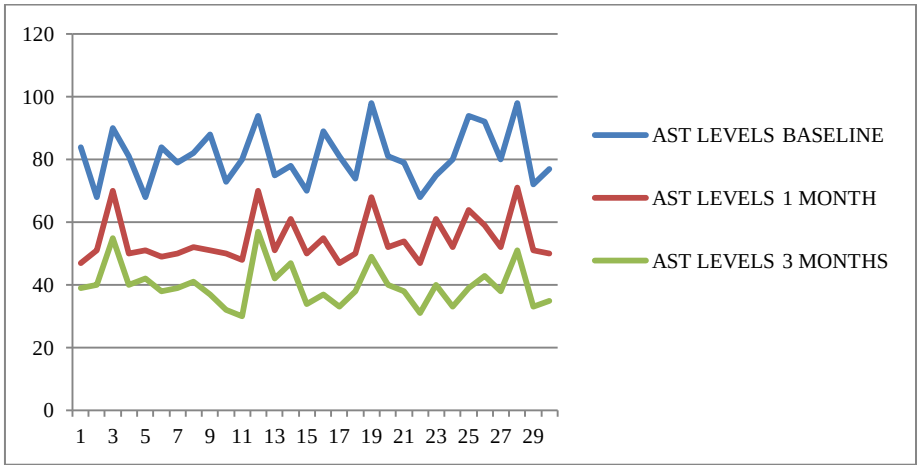


Table 4: Coefficient of correlation between different parameters in group I (Karl Pearson's correlation coefficient test) '*r*'

Parameters	**Baseline**			**1 Month**			**3 Months**		
	"r"	t_{28}	p	"r"	t_{28}	p	"r"	t_{28}	p
GI and AST	0.40	2.30	<0.05	0.14	0.74	>0.05	0.05	0.10	>0.05
PD and AST	0.23	1.25	>0.05	-0.02	-0.10	>0.05	0.06	0.80	>0.05

Table 5: Coefficient of correlation between different parameters in group II (Karl Pearson's correlation coefficient test) '*r*'

Parameters	**Baseline**			**1 Month**			**3 Months**		
	"r"	t_{28}	p	"r"	t_{28}	p	"r"	t_{28}	p
GI and AST	0.16	0.85	>0.05	-0.18	-0.96	>0.05	0.07	0.37	>0.05
PD and AST	0.49	2.97	<0.01	0.34	1.91	>0.05	0.53	3.30	<0.01

IV. Result

Comparison between group I and group II for all the measures of periodontal parameters and AST levels were analyzed by applying Student paired "*t*" test. Karl Pearson's correlation coefficient ("*r*") was calculated among different parameters of periodontal disease and AST concentration. In group I: At baseline, the mean GI was 1.76±0.22, the mean PD was 1.64±0.42 mm, and the mean AST level was 59.9 ± 8.10 Unit/liter [Table 2]. At 1 month, the mean GI was 0.67±0.14, the mean PD was 1.51±0.0.41 mm, and the mean AST level was 46.8 ± 7.38 Unit/liter [Table 2]. At 3 month, the mean GI was 0.44±0.08, the mean PD was 1.38±0.38 mm, and the mean AST level was 24.8 ± 6.73 Unit/liter [table 2, Graphs 1 and 3]. In group II: At baseline, the mean GI was 1.94±0.40, the mean PD was 4.87±0.33, and the mean AST level was 81.0 ± 8.66 Unit/liter [table 3]. At 1 month, the mean GI was 0.72±0.16, the mean PD was 3.06±0.54 mm, and the mean AST level was 54.4 ± 7.37 Unit/liter [table 3]. At 3 month, the mean GI was 0.50±0.12, the mean PD was 2.41±0.37 mm, and the mean AST level was 29.7 ± 6.61 Unit/liter [table 3, graphs 2 and 4].

In the group I: Comparison of clinical and biochemical parameters at baseline and at 3 months exhibited a reduction in GI, PD and AST levels which their found to be statistically significant (*P* value < 0.05) [Table 2].

In the group II: Comparison of clinical and biochemical parameters at baseline and at 3 months exhibited a reduction in GI, PD, and AST levels which their found to be statistically significant (*P* value < 0.05) [Table 3].

V. Discussion

Periodontal disease is a chronic bacterial infection characterized by persistent inflammation, connective tissue breakdown, and alveolar bone destruction.[13] Subjective symptoms are typically mild during the early phase of disease, subjects tend to ignore the condition until more severe symptoms appear, e.g. increased tooth mobility, etc. Periodontal disease may be a major cause of tooth loss.[14]

Traditional diagnostic measures such as periodontal pocket depth, attachment level, plaque index, bleeding on probing, and radiographic assessment of alveolar bone loss are informative to evaluate disease severity but provide few useful determinants of disease activity. It has been a great challenge in periodontology to determine biomarkers for screening and predicting the early onset of disease (prognostic tests) or evaluating the disease activity and the efficacy of therapy (diagnostic tests).[15]

Biochemical tests are used extensively in medicine both in relation to diseases that have an obvious metabolic basis and those in which biochemical changes are a consequence of the disease. In dentistry, these tests are gaining importance in the diagnosis, prognosis, monitoring, and screening of periodontal diseases in which changes in enzymatic activity reflect metabolic changes in gingiva and periodontium in inflammation. These tests have been proposed to assess periodontal disease activity in addition to clinical assessments.[16]

The present study was designed to assess the clinical and biochemical parameters in patients with generalized chronic gingivitis and periodontitis.Thise study population consisted of total 60 subject. the group -I 30 patients with generalized chronic gingivitis and group - II 30 patients with generalized chronic periodontitis with the range of 20-65 years.

At baseline, a decrease in GI was seen in both the groups at 1 month and 3 months which exhibited a statistically significant ($P < 0.05$). The reduction in gingival index is attributed to the effects of SRP. SRP is considered as the gold standard therapy in the treatment of periodontal diseases. SRP improves clinical parameters by decreasing the inflammatory infilterate. This is also in agreement with the studies conducted by **Persson *et al*, Shimada *et al*, and Arora *et al***, which showed that SRP and motivation for oral hygiene helps improve the periodontal health of the subjects.[12]

At baseline, the mean PD in group I was 1.64± 0.42 mm (Table 2). Ideal PD of the gingival sulcus is 0 mm but this can be produced experimentally only in germ frees animals (**Attstrom** and **Caffesse et al**). In a clinically healthy human gingiva, some depth of the sulcus can be found as determined by histologic sections. **Weski** and **Gargiulo *et al***. have reported a depth between 1.5 mm and 0.6 mm, respectively.

At 3 months, the PD in Group I was 1.38 ± 0.38 mm [Table 2]. The minor reduction could be attributed to the effects of SRP. However, the reduction in PD at 3 months as compared to the baseline was found to be statistically significant ($P < 0.05$). Likewise, group II showed a decrease in PD after SRP at 1 month and 3 month which was significant statistically ($P < 0.05$) [Table 3]. This goes in accordance with the studies done by **Arora *et al*, Yoshie *et al*, Shimada *et al***, and **Persson *et al***. who supported that clinical parameters can be improved by SRP.

At baseline, AST level in saliva was 59.9 ± 8.10 U/L in group I [Table 2] and 81.0 ± 8.66 U/L in group II [Table 3]. AST is a cytoplasmic enzyme present in many body tissues with especially pronounced distribution in heart, liver, and skeletal muscle. It is important for the production of various aminoacids and serve as a diagnostic analyte of cellular injury. It is a ubiquitous component of saliva and is detected in periodontal tissue, GCF, enamel pellicle, and saliva.

Their activity can be proved in saliva, within some normal limits, as these enzymes are determined even in blood of healthy persons. However, if there is periodontal tissue destruction, these intracellular enzymes are increasingly being released into the GCF and saliva where their activity can be measured. Due to this, it can be a biochemical marker of the functional condition of periodontal tissues. (**Numabe, Mc Culloch, and Nakashima et al**).

These enzymes are indicators of a higher level of cellular damage and their increased activity in GCF and saliva is a result of their increased release from the damaged cells of soft tissues of periodontium and a reflection of metabolic changes in the inflamed gingiva (**Numabe, Ozmeric, et al**). Studies done by **Kolte, Golub, Cohen, Persson and Page RC et al** have also observed AST levels to be high at diseased sites.

Subject with known history of systemic condition such as heart disease, diabetes, liver, kidney, or salivary gland dysfunction, pregnant or lactating, patients using glucocorticoids, cyclooxygenase inhibitors, bisphosphonates, antibiotics, or immunosuppressant medication during the past 6 months are excluded as that could affect the manifestations of periodontal disease. Also smokers are excluded as it can effect manifestation of periodontal disease in chronic periodontitis and healthy individuals (**Mouzakiti et al 2011 and 2012**).

Previous studies by **Silva, Hanioka, Oringer, Persson, Wong et al** mainly investigated the activities of these enzymes in GCF, which is in a much closer contact with periodontal tissues and due to this, it surely much better reflects the occurrences in them. However, the problem with the GCF is in that the technique of collecting is rather complicated and that in a routine procedure, which possibly might be established, it would be hardly feasible in practice. Contrary to the GCF, there is plenty of saliva, the procedure of its sampling is much

easier and more bearable for the patient and however, the same enzymes as those in the GCF can be detected. Because of the simple and non-invasive method of collection, salivary diagnostic tests hold promise for the future. (***Numabe, Kaufman, Ozmeric* et al**).[12]

Also, improvements in clinical status were noted following periodontal therapy and there was a corresponding decrease in AST levels. So, AST levels may be a sensitive and specific enough to be a useful adjunct in the clinical assessment of periodontal disease, since AST level decrease when periodontal status improves. At baseline, when AST levels were correlated with GI scores, there was a very significant correlation ($P < 0.01$) suggesting that AST level is associated with severity of gingival inflammation. This is in accordance with studies of **Persson** and **Kolte et al**.

At 3 months, following treatment in group I, a positive correlation was present between GI and AST levels [Table 4] but it was insignificant statistically ($P > 0.05$). In clinically healthy gingiva with PD ranging between 0 and 3 mm, small foci of inflammatory cells would be present along the sulcus. This is in accordance with studies conducted by **Page** and **Shroeder et al**, who histologically studied inflammatory changes adjacent to junctional epithelium and gingival sulcus.

At baseline, in group II the correlation between GI and AST levels was positive and statistically significant ($P < 0.05$). This was supported by **Persson *et al***, who suggested that increased periodontal destruction resulted by microbial activity leading to increased AST concentration [Table 5].

At 3 months, in group II a positive correlation was present between GI and AST levels following treatment [Table 5] but it was statistically insignificant. This may be attributed to persistent inflammation in deep pockets post SRP. SRP may be insufficient to completely debride the teeth with deep pockets. Hence, the concentration of AST in saliva would be higher in deep pockets. This was supported by study done by **Arora *et al***.

At baseline, in group I a positive correlation between AST levels and PD was found which was statistically insignificant. This was supported by Kolte *et al*, who has shown that certain inflammatory foci are seen even in healthy gingiva. At 3 months, in group II the correlation between PD and AST levels was found to be a weak correlation ($P > 0.05$). This may be due to residual inflammation and ulceration in the tissues even after SRP. SRP has been found to be insufficient in completely removing the inner lining epithelium. The results go in accordance with studies done by **Persson *et al***, who found a weak correlation between AST values and PD. Fluctuations in AST levels in saliva varied at various sites. This may be due to the active periodontal destruction at the sites. The variations in the AST levels could also be a resultant of the episodic nature of periodontal destruction (**Imrey, Persson, and Page et al**).

Periodontal destruction is associated with the anaerobic microflora resulting in tissue destruction thereby releasing AST in GCF and saliva. Studies conducted by **Kuru et al**. concluded that Porphyromonas gingivalis, Aggregetibacter actinomycetemcomitans, and Prevotella intermedia were significantly higher in AST positive than negative sites. The study thus concludes that AST enzyme levels were significantly elevated in saliva of patients with generalized chronic gingivitis and generalized chronic periodontitis, with higher values recorded in generalized chronic periodontitis patients correlating to the tissue destruction taking place in these conditions. Importance has been given to AST activity in saliva as a diagnostic aid, and studies are still going on in order to confirm whether AST estimation can be used as a specific test to diagnose a periodontitis case and its usefulness in a clinical setting.

Most studies have focused on the levels of these enzymes in the saliva of patients with liver disease as well as periodontitis, because saliva, as a biologically available material, can help diagnose and explain the pathogenesis of some systemic diseases as a diagnostic test (**Zhang et al**., **2016**, **Lee & Wong**, **2009**). However, **Neto et al**, **2011** suggested that the release of enzymes from the tissues covering the salivary glands and blood vessels can be incomplete or that processes such as producing oxygen radicals in the oral cavity can alter the activity of the enzymes.[15] Many enzymes have been used as a biomarker to assess the progression of periodontal diseases. The enzyme AST is one such marker, which has been used as a diagnostic of various human diseases.

The determination of AST levels in serum has been used for many years to identify inflammatory lesions in the heart, liver and kidney, and in cerebrospinal and synovial fluids for lesions in the brain and joints respectively. This enzyme would be expected to pass from the periodontal tissues in the inflammatory exudates into the gingival crevicular fluid (GCF) and saliva. **Deshpande** and **Kohad et al,** observed remarkably higher levels of the AST enzyme in GCF than that in the blood. This rise in GCF enzyme level may be due to the cellular damage, predominantly PMNs at the diseased site, secondly the change in microbial flora at the diseased site may play a contributory role in determination of the enzyme activity.

A very strong association has been demonstrated between periodontal disease active site and the presence of high levels of crevicular fluid AST levels. In ligature induced periodontitis it was found that AST levels correlated with microscopic evidence of tissue destruction. Some studies found that AST correlated with the extent of gingival inflammation and with clinical course. A corcordance between CAL of 2 mm and AST activity of 1200 IU was noted, leading to the acceptance of thise AST value as possible marker of periodontal

disease activity. Although a lot of evidence has accumulated for the use of GCF for diagnosis of periodontitis, thise approach has a demerit as sampling technique is not easy and longer duration is needed foe sample collection in addition it is difficult to obtain GCF from all site of the dentition.

Smith et al. have shown that GCF volume and enzyme activity different among 6 site sampled. Thus it is difficult to present values representative of a subject's oral cavity or even of one tooth. Contrary to the GCF there is plenty of saliva. The procedure of its sampling is much easier and more bearable for the patient, and however, the same enzyme as those in the GCF can be detected. because of the simple and non-invasive method of collection. Salivary diagnostic tests appear to hold promise for the future In a recent paper, **Totan et al**. found a significant increase in salivary enzymatic activities in periodontitis patients, namely AST and alkaline phosphatase and alanine aminotransferase and emphasized their use in monitoring the periodontal disease. A few studies analyzed the levels of AST in GCF and saliva before and after periodontal treatment in periodontitis patients. Improvements in clinical status were noted following periodontal therapy and there was a corresponding decrease in AST levels.[17]

VI. Conclusion

The following conclusions were drawn from the present study:

1) Salivary aspartate aminotransferase enzyme was detected in all samples.

2) Satistically significant difference was found between aspartate aminotransferase concentration in chronic generalized periodontitis and chronic generalized gingivitis in patients at baseline and after scaling and root planing at follow-up 1month and 3rd months. ($p<0.05$).

3) Aspartate aminotransferase concentration were significant higher in chronic periodontitis patients (81.0 ± 8.66) than that in chronic gingivitis patients (59.9 ± 8.10).

4) Statistically significant difference was found in clinical parameters in chronic periodontitis and chronic gingivitis patients at baseline and after scaling and root planing at follow-up 1 month and 3 months ($p<0.05$).

5) At baseline, in group II the correlation between GI and AST levels was positive and statistically significant ($P < 0.05$).

6) At 3 months, in group II a positive correlation was present between PD and AST levels following treatment [Table 5] but it was statistically insignificant. This may be attributed to persistent inflammation in deep pockets post SRP. SRP may be insufficient to completely debride the teeth with deep pockets.

7) At baseline, in group I a positive correlation between AST levels and PD was found which was statistically insignificant.

8) At 3 months, in group II the correlation between PD and AST levels was found to be a weak correlation ($P > 0.05$). This may be due to residual inflammation and ulceration in the tissues even after SRP. SRP has been found to be insufficient and incompletely removing the inner lining epithelium.

The present study suggest that AST might be useful for measurement of periodontal disease activity, possibly due to the fact that salivary AST may reflect tissue necrosis and also few studies have reported that after periodontal treatment the activity of AST enzyme was decreased, which was probably measuring tissue repair, hence it can be useful in monitoring treatment outcome.[12]

The finding of thise study indicates that measuring salivary aspartate aminotransferase concentration can be considered an inflammatory biomarker for measuring the severity of periodontal inflammation. studies with small sample size and for longer need to be conducted before and after periodontal treatment to confirm the finding of the present study & for future research.

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